A Dissertation on

STUDY OF INTRAVENTRICULAR CONDUCTION DISTURBANCES AND LEFT VENTRICULAR HYPERTROPHY IN SYSTEMIC HYPERTENSION

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CERTIFICATE

This is to certify that the dissertation entitled **"STUDY OF INTRAVENTRICULAR CONDUCTION DISTURBANCES AND LEFT VENTRICULAR HYPERTROPHY IN PATIENTS WITH HYPERTENSION IN GOVERNMENT ROYAPETTAH HOSPITAL"** is a bonafide work done by **DR. P.SENTHUR NAMBI ,** Post graduate student, Department of Internal Medicine, Kilpauk Medical College, Chennai-600 010, under my direct guidance and supervision in fulfillment of the regulations of **THE TAMIL NADU DR.MGR MEDICAL UNIVERSITY** for the award of MD Degree Branch I, Part II (General Medicine) during the academic period from May 2005 to March 2008.

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CONTENTS

INTRODUCTION

Hypertension is one of the leading causes of morbidity and mortality in the world and will increase in world wide importance as a public health problem by the year 2020 [1]. It is one of the most common diseases which is easily detectable, readily treatable, often leading to fatal complications if left untreated.

Hypertension is a powerful risk factor that increases the likelihood of developing a wide variety of cardiovascular diseases like cerebrovascular accident, coronary artery disease, heart failure, renal insufficiency and peripheral vascular disease. [2]

Hypertension causes an increase in left ventricular mass and fibrous tissue resulting in increased stiffness of the left ventricle leading possibly to reduced coronary reserve, silent myocardial ischaemia, abnormal electrophysiological properties of hypertrophied myocytes and conduction disturbances. Electrocardiogram is one of the tests recommended by the Seventh Joint National Committee of hypertension for the initial evaluation of a hypertensive patient [3]. Not only is the Electrocardiogram useful in documenting previously undetected myocardial infarction, myocardial ischaemia and conduction disturbances, but also it is the least expensive and possibly the most cost effective way to diagnose or exclude left ventricular hypertrophy.

AIM OF THE STUDY

To study the incidence of intraventricular conduction disturbances in patients with systemic hypertension.

To identify the most common type of intraventricular conduction disturbance.

To study the incidence of electrocardiographic evidence of left ventricular hypertrophy with or without strain in patients with systemic hypertension.

REVIEW OF THE LITERATURE

ANATOMY OF THE CARDIAC CONDUCTION SYSTEM

ELECTROPHYSIOLOGY OF THE CONDUCTION SYSTEM

HYPERTENSIVE HEART DISEASE

INTRAVENTRICULAR CONDUCTION DISTURBANCES

1. CARDIAC CONDUCTION SYSTEM

The structures that make up the cardiac conduction system are

- The SinoAtrial node (SA node)
- Internodal Pathways
- AV node
- Bundle of His
- Right and Left Bundle branch
- Anterior and Posterior fascicle of the Left Bundle Branch
- Purkinje Fibers

The SA node is the pacemaker of the heart. Impulses generated in the SA node pass through the atrial pathways to the AV node, then through this node to the bundle of His and through the branches of bundle of His and finally via the Purkinje system to the ventricles.

ANATOMY [4,5]

S A NODE

The SA node is the pacemaker of the heart. It is a small flattened ellipsoid strip of specialised muscle about 3 mm wide, 15 mm long and 1 mm thick

It is situated in the antero superior wall of the right atrium at the upper end of sulcus terminals immediately anterior and lateral to the opening of the superior vena cava. The fibres of SA node are 3-5 microns in diameter, in contrast to the diameter of 15 to 20 microns of the surrounding atrial muscle fibers.

INTERNODAL PATHWAYS

These are the bundles of atrial fibers that contains the specialised Purkinje type fibers conducting the impulses at a higher velocity compared to the surrounding atrial muscle fibers. They connect the SA node to the AV node and there are three such bundles.

They are

- Anterior internodal tract of BACHMANN
- Middle internodal tract of WENCKEBACH
- Posterior inter nodal tract of THOREL

ANTERIOR INTER NODAL TRACT

It leaves the anterior margin of SA node and passes anterior to the superior venacava to enter the crest of AV node.

MIDDLE INTERNODAL TRACT

It leaves the posterior margin of the SA node and passes posterior to the superior venacava, then descends in the inter atrial septum and merges with the fibers of the anterior inter nodal tract to enter the crest of AV node.

POSTERIOR INTERNODAL TRACT

Leaves the posterior margin of SA node and runs with the crista terminalis, then curves through the valve of Inferior venacava to enter the posterior margin of AV node.

ATRIO VENTRICULAR NODE (AV NODE)

It is smaller than the SA node and is situated in the right atrial side of the interatrial septum, just above the opening of the coronary sinus. It is continuos with the bundle of His. The bundle then passes in the membranous part of the interventricular septum and divides into right and left bundle branches on either side of the muscular septum.

RIGHT BUNDLE BRANCH

It travels down the right side of inter ventricular septum to reach the anterior wall of the right ventricle and then enters the base of the anterior papillary muscle.

LEFT BUNDLE BRANCH

It is a broad band which usually divides into two divisions namely the left anterior fascicle and left posterior fascicle. These divisions should be thought of as groups of interconnecting fascicles as opposed to individual, insulated, free running divisions.

The branches and fascicles run subendocardially down either side of the septum and come into contact with the Purkinje system.

PURKINJE SYSTEM

The Purkinje system consists of very large fibers, even larger than the normal ventricular fibers. The rapid transmission of impulses by Purkinje system is probably caused by increased numbers of nexuses between the successive cardiac cells that make up the Purkinje fibers. They have very few myofibrils and barely contract during the course of impulse conduction.

The terminal Purkinje fibers penetrate about one third of the way into the muscle mass to terminate on the muscle fibers.

BLOOD SUPPLY OF THE CONDUCTION SYSTEM

The vascular supply of the SA node is through a small artery that arises about two centimeters from the ostium of the right coronary artery in approximately sixty percent of the population and from the proximal part of the left circumflex artery in forty percent of the population.

In ninety percent of the population the blood supply of AV node is through the AV nodal branch of the right coronary artery. In the remaining ten percent, the blood supply is through a branch of the left circumflex artery.

The bundle of His and the interventricular septum have a dual blood supply which arise from the posterior descending branch of right coronary artery and septal perforating branch of left anterior descending artery.

The right bundle branch and left posterior fascicle of the left bundle branch have a dual blood supply from the left anterior descending and right coronary arteries whereas the left anterior fascicle of the left bundle branch is supplied by the septal perforators originating from the left anterior descending artery.

INNERVATION OF THE HEART

Parasympathetic nerves reach the heart via the vagus. Sympathetic nerves are derived from the third to fifth thoracic segments of the spinal cord. Both parasympathetic and sympathetic nerves form the superficial and deep cardiac plexuses, the branches of which run along the coronary arteries to reach the myocardium.

The superficial cardiac plexus is situated below the arch of aorta in front of the right pulmonary artery whereas the deep cardiac plexus is situated in front of the bifurcation of the trachea, above the point of division of the pulmonary trunk and posterior to the aortic arch.

Sympathetic fibers are cardio acceleratory and on stimulation they increase the heart rate and also dilate the coronary arteries while the parasympathetic fibers are concerned with the slowing of the heart rate and constriction of coronary arteries.

The SA node develops from structures on the right side of the embryo and the AV node from structures on the left. This is why in the adult the right vagus is distributed mainly to the SA node and the left vagus mainly to the AV node. Similarly, the sympathetic innervation on the right side is distributed primarily to the SA node and the sympathetic innervation on the left side primarily to the

AV node. Noradrenergic fibers are epicardial, whereas the vagal fibers are endocardial. However, connections exist for reciprocal inhibitory effects of the sympathetic and parasympathetic innervation of the heart on each other.

ELECTROPHYSIOLOGY OF THE CONDUCTION SYSTEM [6]

Along with nerve, skeletal muscle, and smooth muscle, heart muscle is one of the excitable tissues of the body. It shares many bioelectrical properties with other excitable tissues but has unique electrical features as well. The electrical activity of the heart is responsible for coordinating the sequence of cardiac activation and contraction. Understanding the normal electrocardiogram requires a substantial knowledge of normal cardiac electrophysiology.

ION CHANNELS

Ion Channels are components of cellular membranes that permit movement of ions across the hydrophobic barriers of the cell membrane. The channels are thought to be membrane spanning proteins that contain water so that hydrated ions cross the membrane. The channels are selective to certain ions, sodium channel preferentially conducts sodium ions and is less permeable to potassium or calcium ions. Channels also have gating mechanisms, that is they may be open or closed. Depending on the chemicals (agonists) in the channel or the transmembrane voltage field, the degree to which the channel opens may vary. Once open, channels have characteristic open times, 1 millisecond for sodium

channels and more than 100 milli second for calcium channels. Each channel seems to operate independently that is the probability that a channel will be open or closed is not influenced by the state of neighbouring channels.

CARDIAC RESTING MEMBRANE POTENTIAL AND ACTION POTENTIALS

The resting membrane potential of individual mammalian cardiac muscle cells is about – 90 milli volts. Stimulation produces a propagated action potential that is responsible for initiating contraction. Depolarisation proceeds rapidly and an overshoot is present as in skeletal muscle and nerve but this is followed by a plateau phase before the membrane potential returns to the baseline. Depolarisation lasts for about two milliseconds, but the plateau phase and repolarisation lasts for two hundred milliseconds or more.

As in other excitable tissues, changes in the external potassium ion concentration affect the resting membrane potential of cardiac muscle, whereas changes in the external sodium ion concentration affect the magnitude of the action potential.

The cardiac action potential has five phases

Phase

0 - Initial rapid depolarisation

1 - Initial rapid repolarisation

2 - Plateau

3 - Final repolarisation

4 - Diastolic Interval

The initial rapid depolarisation and the overshoot (Phase 0) are due to the opening of voltage gated sodium ion channels.

The initial rapid repolarization (Phase 1) is due to the closure of sodium ion channels.

The subsequent prolonged plateau (Phase 2) is due to a slower but prolonged opening of the voltage gated calcium ion channels.

Final repolarisation (Phase 3) to the resting membrane potential (Phase 4) is due to the closure of the calcium ion channels and potassium ion efflux through the three types of potassium ion channels.

PACEMAKER POTENTIAL

Rhythmically discharging cells have a membrane potential that after each impulse declines to the firing level. Thus this prepotential or pacemaker potential triggers the next impulse. The calcium current due to the opening of

transient calcium channels completes the prepotential and the calcium current due to opening of the long lasting calcium channels produces the impulse .

The action potentials in the SA and AV Nodes are largely due to calcium ions with little contribution by sodium ion influx. Consequently there is no sharp, rapid depolarising spike before the plateau, as in other parts of the conduction system. In addition, prepotentials are normally prominent only in SA and AV nodes. However, there are latent pacemakers in other portions of the conduction system that can take over when the SA and AV nodes are depressed. Atrial and ventricular muscle fibres do not have prepotentials and they discharge spontaneously only when injured or abnormal.

SPREAD OF CARDIAC EXCITATION

Depolarisation initiated in the SA node spreads radially through the atria, then converges on the AV Node. Atrial depolarisation is complete in about 0.1 second. Since conduction in the AV Node is slow, there is a delay of about 0.1 second (AV nodal delay) before excitation spreads to the ventricles. From the top of the septum, the wave of depolarisation spreads in the rapidly conducting Purkinje fibres to all parts of the ventricles in 0.08 – 0.1 seconds.

In humans, depolarisation of the ventricular muscle starts at the left side of the interventricular septum and moves first to the right across the midportion of the

septum. The wave of depolarisation then spreads down the septum to the apex of the heart. It returns along the ventricular walls to the atrioventricular groove, proceeding from the endocardial to epicardial surface. The last parts of the heart to be depolarised are the postero basal portion of the left ventricle, the pulmonary conus and the uppermost portion of the septum.

VELOCITY OF CONDUCTION IN CARDIAC TISSUE

Tissue	Conduction Rate (Metres / Second)
SA Node	0.05
Atrial Pathways	1
AV Node	0.05
Bundle of His	1
Purkinje System	4
Ventricular Muscle	1

There are anatomic reasons for the slow conduction in the sinus node and AV node. The cell to cell connections between the sinus node P cells or the AV node N Cells and their neighbouring cells are sparse and primitive. This contributes to the slow spread of excitation in these structures. The Slow propagation of impulse in the sinus node and AV node permits reentrant excitation to occur in very small areas despite the long refractory period found in cardiac muscle.

INTRAVENTRICULAR CONDUCTION DISTURBANCES

Of the various forms of intraventricular block, bundle branch block is most common and best recognized

The various intraventricular conduction disturbances are

- Right Bundle Branch Block (RBBB)
- Left Bundle Branch Block (RBBB)
- Left Anterior Fasicular Block (LAFB)
- Left Posterior Fasicular Block (LPFB)
- Bifasicular block
- Trifasicular block
- Non Specific intra ventricular conduction disturbances

BASIC PRINCIPLES[7]

If one of the branches of the bundle of His is blocked by disease the impulse travels down the branch to the other ventricle first. Having activated this ventricle the impulse spreads through the septum to the ventricle on the side of the block and in turn activates it. Thus the ventricles are activated one after the other instead of simultaneously. This results in prolongation of the QRS interval and the ST segments slopes off in the direction opposite to the main QRS deflection in cases of bundle branch block.

Bundle branch blocks are recognized by their prolongation of QRS interval while fascicular blocks are associated with normal QRS interval

In the Framingham study, it was concluded that people with bundle branch block were more likely to have or to subsequently develop cardiovascular manifestation, especially in men with LBBB.[8]

Bundle branch Block can be produced by involvement of the fibers in the branch or in the His bundle itself by many pathological process. Very commonly it is due to either ischemia or fibrosis associated with ventricular hypertrophy as in hypertension. Same conditions can also be responsible for other types of intraventricular conduction disturbances in the form of fascicular blocks. In most patients, unifasicular, bifasicular or trifasicular diseases is secondary to readily apparent organic heart disease.

Histologically in hypertensive heart disease, moderate fibrosis, fatty infiltration and arteriosclerosis were present in the internodal pathways. There was partial or complete fibroelastic disruption of the bundle branches.[9]

DIAGNOSTIC CRITERIA OF CONDUCTION DEFECTS

1. RIGHT BUNDLE BRANCH BLOCK (RBBB) [10]

Right bundle branch block is a variety of unifascicular block. In this unifascicular block, impulses are delivered by the left bundle branch and this crosses the septum to activate the right ventricle. This produces QRS widening.

DIAGNOSTIC CRITERIA FOR RBBB

1. QRS complex $\geq$ 0.12 sec
2. rSR' (or) "M" pattern of QRS in V_{1-3}, sometimes wide "R" (or) qR pattern present
3. Deep, wide, slurred "S" wave in Lead I, aVL, V_{4-6}
4. Secondary ST-T changes in V_{1-3}

CLINICAL SIGNIFICANCE

Anatomically, right bundle branch is a long microscopic structure and consequently a minute lesion may produce a delay in the transmission of an impulse along its path.

In Hess and Lamb's Study, RBBB was found in 0.2 percent of otherwise normal males.[11]

Jeong et al reported the incidence of RBBB in normal persons of greater than forty years of age to be 1.2 percent.[12]

Conditions associated with RBBB are coronary artery disease, hypertension, valvular heart disease, congenital heart diease, myocarditis.

Hardarson et al reported that in people less than sixty years of age with RBBB, there was positive correlation to hypertension.[13]

LEFT BUNDLE BRANCH BLOCK (LBBB) [14]

In this condition the left ventricle is activated after the right ventricle and the initial septal activation begins from right to left instead of the normal left to right.

DIAGNOSTIC CRITERIA

1. QRS complex $\geq$ 0.12 sec
2. Absence of septal 'q' waves in lead I, aVL, V_{4-6}
3. rSR', "M" pattern (or) Broad "R" waves in lead I, aVL and V_{4-6}
4. Broad "Q-s (or) "rS" waves in leads V_{1-3}
5. Secondary ST-T changes in LeadI, aVL, V_{4-6}

CLINICAL SIGNIFICANCE

The main left bundle divides closer to its origin than the right bundle, so that a relatively smaller area of the left bundle is vulnerable to interference. A larger lesion would therefore be required to block conduction in the main left bundle than would be required to block conduction along the right bundle.[15] Hence RBBB is more common than LBBB.

Jeong et al in his study of 14540 persons, reported the incidence of LBBB to be 0.3 percent.[12]

Isolated LBBB is associated with an increased risk of developing overt cardiovascular disease, with increased cardiac mortality.[16]

Among patients with LBBB, those with left axis deviation have a greater incidence of myocardial dysfunction, more advanced conduction disease and greater cardiovascular mortality than those with normal axis.[17]

LEFT ANTERIOR FASCICULAR BLOCK(LAFB) [18]

Since the impulse conduction is via the posterior division it is directed superiorly and to the left, a marked left axis deviation is observed in LAFB.

DIAGNOSTIC CRITERIA FOR LAFB

1. Narrow QRS complex < 0.10 sec
2. Left axis deviation -45◦ to -90◦ (usually -60◦)
3. "qR" complex in lead I, aVL, V_{5-6}; "rS" complex in lead II, III, aVF
4. Late intrinsicoid deflection in aVL > 0.045 sec
5. Increased QRS voltage in Limb leads
6. Persistent S (V5, V6)

CLINICAL SIGNIFICANCE

Since both the right bundle and the left anterior fascicle share vulnerability to injury, it is not too surprising that RBBB and LAFB occur together with greater frequency.[19] In order to assess its prognosis, it is necessary to evaluate the ECG findings in the context of underlying disease process.[20]

Patients without underlying heart disease are probably not at increased risk of heart block or sudden death.[21,22] Mac Alpin et al in his review of 26000 ECG observed findings consistent with LAFB in 0.5% of tracings.[23]

LEFT POSTERIOR FASCICULAR BLOCK (LPFB)

DIAGNOSTIC CRITERIA FOR LPFB

1. Narrow QRS compex ≤ 0.10 sec
2. Right axis deviation – QRS axis ≥ 110◦

3. “rS” complex in L_I,aV_L
4. “qR” complex in Leads II, III, aV_F
5. Absence of RVH or other causes for right axis deviation.

CLINICAL SIGNIFICANCE

Left posterior fascicle is the least vulnerable segment of the whole intraventricular conduction system. If it is involved then it is prudent that the lesions are so widespread in the heart or in the conduction system.[24]

BIFASCULAR BLOCK

Refers to the block of two fascicles of the trifasicular conduction system. The combinations producing bifascular block are

Predivisional block of LBB

RBBB + LAFB

RBBB + LPFB

RBBB with LAFB is diagnosed when V1 demonstrates RBBB and frontal plane leads demonstrate left axis deviation.

CLINICAL SIGNIFICANCE

Rosenbaum et al reported the development of complete heart block in seventy percent patients with RBBB and LPFB.[25]

Barret et al reported the development of complete heart block in nineteen percent of patients with RBBB and LAFB.[26]

TRIFASICULAR BLOCK

It requires an ECG Pattern of Bifasicular block and evidence of prolonged AV conduction (PR interval greater than twenty millisecond). Forty percent of patients with trifasicular block progress to complete heart block.

NON SPECIFIC INTRAVENTRICULAR CONDUCTION DISTURBANCES [27]

Block that occurs distal to the main division of the bundle branches has been referred to under a large variety of names such as focal block, arborisation block, reticular block, post infarction block, parietal block.

Several factors, including increased potassium ions, acidosis, injection of contrast media in coronary artery, diffuse myocardial fibrosis, large scars, infarcted areas may be responsible for this widening of the QRS complex which does not fulfill the criteria for RBBB or LBBB.

The presence of normal q wave supports conduction delay in the peripheral purkinje fibres or in the myocardial fibers themselves as the cause of prolongation of QRS.

Features

1. QRS is prolonged (> 0.10 second)
2. Absence of characteristic precordial lead QRS pattern.

HYPERTENSIVE HEART DISEASE

THE JOINT NATIONAL COMMITTEE (JNC) CLASSIFICATION of HYPERTENSION [3]

BLOOD PRESSURE CATEGORY	Blood Pressure Range (Systolic, Diastolic, mm Hg)
Normal	< 120 and < 80
Prehypertension	120-139 or 80-89
Stage 1	140-159 or 90-99
Stage 2	≥ 160 or ≥ 100

In the scheme of classification and stratification of hypertension in the seventh report of the Joint National Committee on prevention, detection, evaluation and treatment of high blood pressure, the stages represent increasing relative risk as blood pressure rises.

The heart is one of the organs most affected as a consequence of hypertension. The spectrum of hypertensive heart disease includes

1. Left ventricular Hypertrophy (LVH)
2. Left ventricular systolic dysfunction
3. Left ventricular diastolic dysfunction
4. Heart failure[28]

MORPHOLOGICAL PATTERNS OF LVH[29]

Concentric LVH

In this form of hypertrophy, myofibrillar units are added in parallel to offset the effects of increased afterload; the result is a uniform thickening of the left ventricle at the expense of the chamber volume.

Early in the course of concentric LVH, subtle evidence of ventricular dysfunction is seen in the form of decreased fractional shortening of cardiac myofibrils,[30] impaired diastolic relaxation and reduced coronary blood flow reserve. Later in life, concentric LVH leads to more advanced left ventricular diastolic dysfunction and eventually predisposes to systolic heart failure dysrhythmias, intraventricular conduction blocks and sudden cardiac death.

ECCENTRIC LVH

In this form of hypertrophy, myofibrillar units are added in sequence leading to an enlarged ventricular chamber with increased end diastolic volume, normal end diastolic pressures and enhanced contractile efficiency.

The ratio of left ventricular wall thickness to the left ventricular chamber radius is increased in concentric hypertrophy whereas in eccentric hypertrophy this ratio is normal.

Patterns of LVH varies with the type of hypertension. In combined systolic and diastolic hypertension, eccentric hypertrophy is more common than concentric hypertrophy,[31,32] whereas in isolated systolic hypertension concentric hypertrophy is more common.

In the pathogenesis of LVH, recent studies has shown that diastolic dysfunction precedes myocardial hypertrophy.[33] Diastolic dysfunction was the earliest cardiac abnormality in hypertension appearing even without an increase in left ventricular mass. It is determined by an increase in left ventricular end diastolic pressure and was noted in half of three hundred patients evaluated by M-mode echocardiography.[34]

RENIN – ANGIO TENSIN SYSTEM – ITS ROLE IN LVH

It is suggested that chronic activation of Renin – Angiotensin system is associated with the development of myocardial fibrosis. In this regard the role of "RENIN- ANGIOTENSIN, BRADYKININ – PROSTAGLANDIN SYSTEM" in the regulation of myocardial collagen metabolism is well documented.

The major structural proteins of the myocardial collagen matrix are the fibrillar collagens type I and type II . The concentration of type I collagen determines the stiffness of the myocardium. Collagen synthesis occurs in the myocardium in response to the Angiotensin II stimulation of fibroblasts, while prostaglandin E_2 inhibits the collagen synthesis. Collagen degradation is mainly effected through the enzyme MMP – I (Matrix Metalloproteinase) – a key enzyme in collagen degradation.

The direct effects of Angiotensin II on cardiac fibroblasts require the presence of specific receptors on these cells. The presence of such Angiotensin – II receptors namely type I (AT_1) and Type II (AT_2) receptors on cardiac fibroblasts and human myocardium has been shown . AT_1 receptor is predominantly involved in the mediation of collagen synthesis where as MMP – I inhibition by Angiotensin II may be mediated through AT_2 Receptor.

Chronic inhibition of cardiac angiotensin converting enzyme would induce both inhibition of myocardial angitensin II generation and elevation of myocardial PGE_2 levels.

This may explain why the ACE inhibitor Lisinopril has been shown to be a powerful agent in regressing myocardial fibrosis in hypertensive heart disease.

EFFECTS OF LVH

Pathological cardiac hypertrophy is associated with adverse effects on cardiac muscle such as altered diastolic function, increased collagen content, alterations in myocardial perfusion, abnormal electrophysiological properties and decreased contractility.

LVH is associated with intimal hyperplasia of epicardial coronary vessels, increased coronary vascular resistance, decreased coronary flow reserve, increased severity and frequency of ventricular dysrhythmia and reduced diastolic relaxation. At the extreme, diastolic dysfunction and restrictive cardiomyopathy results clinically in “Flash Pulmonary edema” despite a normal ejection fraction on echocardiography and it carries a poor prognosis.[35,36]

EFFECT OF LVH ON CORONARY CIRCULATION

Patients with hypertrophied ventricles often exhibit signs and symptoms of myocardial ischaemia in the absence of coronary artery disease. This has been proved by many angiographic studies in hypertensive patients who complained of anginal pain.

Studies in animals and man indicate that the major conduit epicardial coronary vessels are enlarged in hypertrophied ventricles. This is however less when compared to the amount of increase in ventricular mass.

Many studies have demonstrated a decrease in capillary density in the subendocardial layers of hypertrophied ventricles secondary to pressure overload. The physiological function of coronary collateral circulation is not enhanced by the pressure induced cardiac hypertrophy.

Both volume and pressure induced hypertrophy in patients of all ages are associated with moderately decreased coronary reserve. The impairment of coronary reserve in hypertension is based on structural and functional alterations in the coronary vessels.

Coronary reserve may also be impaired due to increased metabolic demands under baseline conditions. These changes result in abnormal perfusion of the hypertrophied ventricles and if severe enough may lead to intermittent episodes of subendocardial ischaemia and eventually to diffuse subendocardial fibrosis.

CRITERIA FOR LEFT VENTRICULAR HYPERTROPHY

ELECTROCARDIOGRAPHY [37]

Sokolow – Lyon index (SV1 + R V5 or 6) > 3.5 mv

(or)

(RV 5 or RV 6) > 2.6 mv

Cornell Voltage index (SV 3 + Ravl) > 2.8 mv in males

(SV 3 + Ravl) > 2.0 mv in females

Cornell Product (SV 3 + Ravl) x QRS duration > 244 V.S

The Romhilt and Estes point score system [38]

Increased QRS magnitude	=	3 points
ST-T abnormalities	=	3 points
Left atrial enlargement	=	3 points
Left axis deviation	=	2 points
Increased ventricular activation time	=	1 point

A score of 5 points or more indicates left ventricular hypertrophy and correlates with increased ventricular mass.

Applying the Sokolow – Lyon Index, the median sensitivity and specificity for detecting left ventricular hypertrophy is thirty one percent and eighty nine percent respectively.[39]

THE LEFT VENTRICULAR STRAIN PATTERN

The classic strain pattern is downslopping ST Segment depression in the lateral precordial leads (V4-V6). It correlates reasonably with echocardiography for LVH and is the strongest ECG marker of increased cardiovascular morbidity and mortality.[40]

PROGNOSTIC SIGNIFICANCE OF LVH

LVH is often considered the "haemoglobin A_{1c} of BP" as it is an objective measure of both the severity and the duration of elevations in BP. In the Framingham heart study, ECG evidence of LVH was associated with three fold increase in the incidence of cardiovascular events.[41]

ECG	Relative Risk for Cardiovascular events (Range)[42]
LVH	2.0 (women) to 2.7 (men)
LVH with strain	2.5 (women) to 3.8 (men)

Okin et al showed that the presence of LVH is predictive of a 2.5 greater risk for all cause mortality.[43] LVH was the most powerful of any of the traditional

cardiovascular risk factors in predicting not only coronary artery disease but also heart failure and stroke.[44]

Impaired left ventricular performance occurred more often in hypertensive patients who had LVH than in those without this condition. The impaired LV performance may contribute to the high incidence of cardiovascular events in this population.[45]

Fahy et al observed that the electrocardiographic signs of LVH preceded the development of LBBB in twenty six percent of patients with LBBB.[16]

Reduction of LVH by antihypertensive therapy may predict a lesser risk for subsequent cardiovascular morbidity.[44] Hence hypertensive patients with LVH should be treated vigorously.

PREVALENCE OF LVH

In patients with stage 1 hypertension, the prevalence of LVH varies between fifteen to thirty percent and in patients with stage 2 hypertension, the prevalence varies between thirty to fifty percent.[31]

LVH is seen by ECG in five to ten percent of hypertensives but is found by echocardiography in about 30% of all hypertensive adults[46]. Using stringent criteria, the sensitivity of ECG for LVH can rise upto twenty percent.

DETERMINANTS OF LVH[47,48]

AGE

Prevalence of LVH increases with age. Data from Framingham cohort has demonstrated a prevalence of LVH of sixteen percent for men and nineteen percent for women, which increases after the age of seventy to thirty percent for men and forty percent for women.

SEX

LVH is more prevalent in women[49]

RACE

Black race is more prone for LVH [50]

BLOOD PRESSURE

BP is the most important determinant of LVH whether hypertension is established by office, home or ambulatory BP Monitoring.[51]

OBESITY

LVH is more prevalent in obese individuals.

SALT INTAKE

A number of studies have shown that salt intake is a strong determinant of LVH.

MATERIALS AND METHODS

SETTING

This study was conducted in the Government Royapettah Hospital during the period from FEBRUARY 2007 to SEPTEMBER 2007. The patients registered in the hypertension clinic were taken up for the study.

STUDY POPULATION

Two hundred and fifty patients were selected randomly and included in the study group and their age ranged from thirty to eighty years.

METHOD

A detailed history and a thorough clinical examination was done in all patients.

The average of two blood pressure readings recorded five minutes apart in sitting position at two visits separated by a week were taken into account.

Standard twelve lead ECG was recorded. Those patients with abnormal ECG were followed up a week later with another ECG. Only patients with persistent ECG abnormalities were taken up for analysis.

Intraventricular conduction disturbances and LVH were documented using standard ECG criteria as mentioned above.

Routine blood pressure, urine analysis and chest x-ray were taken in all patients

INCLUSION CRITERIA

Only patients with history of hypertension for more than a year were taken up for the study.

EXCLUSION CRITERIA

Patients with clinical and ECG evidence of coronary artery disease, valvular heart disease and congenital heart disease were excluded from the study.

Patients with history of diabetes,chronic obstructive airway disease were excluded from the study.

Patients with secondary hypertension were excluded from the study.

OBSERVATION

The Electrocardiograms of two hundred and fifty patients with persistent hypertension were analysed using the electrocardiographic criteria for intraventricular conduction defects and left ventricular hypertrophy as described above. The study group consisted of one hundred and thirty male patients and one hundred and twenty female patients. One hundred and eighteen patients were of lesser than 50 yrs old and one hundred and thirty two patients were of greater than 50 years of age.

1. **NCIDENCE OF IVCD AND LVH IN HYPERTENSIVES**

ECG	NUMBER OF PATIENTS	PERCENTAGE
IVCD	25	10%
LVH (WITH OR WITHOUT STRAIN)	43	17.2%
NORMAL ECG	182	72.8%
TOTAL	250	100%

In our study group of 250 patients, 43 patients (17.2%) had LVH with or without strain and 25 patients (10%) had intraventricular conduction disturbances. 182 patients (72.8%) had normal ECG.

2. **INCIDENCE OF IVCD IN HYPERTENSIVES**

IVCD TYPE	NUMBER OF PATIENTS	PERCENTAGE
RBBB	6	2.4%
LBBB	1	0.4%
LAFB	18	7.2%
TOTAL	25	10%

IVCD was observed in 25 patients (10%) of our study group. LAFB was observed in 18 patients (7.2%), RBBB was observed in 6 patients (2.4%). Only one patient(0.4%) had LBBB. LPFB was not observed in our study group .

LAFB was the most common IVCD, followed by RBBB and LBBB respectively.

3. **SEX WISE DISTRIBUTION OF IVCD**

IVCD TYPE	NUMBER OF MALE PATIENTS	NUMBER OF FEMALE PATIENTS	TOTAL
RBBB	3	3	6
LBBB	1	0	1
LAFB	10	8	18
TOTAL	14	11	25

The incidence of IVCD in male hypertensives was 10.1% and it was 9.1% in female hypertensives. Out of the 18 patients with LAFB, 10 patients were male and 8 patients were female. Out of the 6 patients with RBBB, 3 patients were male and 3 patients were female. LBBB was observed in a male patient.

The number of male and female patients showing IVCD was 14 and 11 respectively .

4. AGE WISE DISTRIBUTION OF IVCD

IVCD TYPE	NUMBER OF PATIENTS ≥ 50 YEARS OF AGE	NUMBER OF PATIENTS < 50 YEARS OF AGE	TOTAL
RBBB	4	2	6
LBBB	1	0	1
LAFB	12	6	18
TOTAL	17	8	25

The incidence of IVCD in patients greater than 50 years of age was 12.3% and it was 6.7% in patients lesser than 50 years of age. Out of the 18 patients with LAFB, 12 patients belonged to the greater than 50 years age group and 6 patients were in lesser than 50 years age group. 4 patients with RBBB were greater than 50 years old and 2 patients were less than 50 years of age .

5. CORRELATION BETWEEN IVCD AND DIASTOLIC BP

IVCD TYPE	PATIENTS WITH DIASTOLIC BP		TOTAL
	< 110 MM Hg	≥110 mmHg	
RBBB	2	4	6
LBBB	0	1	1
LAFB	5	13	18
TOTAL	7	18	25

Out of 18 patients with LAFB, 13 patients had diastolic BP greater than 110 mmHg and 5 patients had diastolic BP less than 110 mm Hg. Out of the 6 patients with RBBB, 4 patients had diastolic BP greater than 110 mm Hg and 2 patients had diastolic BP less than 110 mm Hg. The one patient with LBBB had diastolic BP greater than 110 mm Hg .

18 patients with diastolic BP ≥ mmHg had IVCD and 7 patients with diastolic BP ≤ 110 mm Hg had IVCD.

6. INCIDENCE OF LVH IN HYPERTENSIVES

LVH	**NUMBER OF PATIENTS**	**PERCENTAGE**
WITHOUT STRAIN	31	12.4%
WITH STRAIN	12	4.8%
TOTAL	43	17.2%

Out of the 250 patients in the study group, 43 patients (17.2%) had ECG evidence of LVH. Out of these 43 patients with LVH, 31 patients (12.4%) had ECG evidence of LVH without strain and 12 patients (4.8%) had ECG evidence of LVH with Strain.

7. SEX WISE DISTRIBUTION OF LVH

LVH	NUMBER OF MALES	NUMBER OF FEMALES	TOTAL
WITHOUT STRAIN	14	17	31
WITH STRAIN	6	6	12
TOTAL	20	23	43

The incidence of LVH in male hypertensives was 15.3% and it was 19.1% in female hypertensives. Out of the 31 patients with LVH without strain, 17 patients were female and 14 patients were male. Out of the 12 patients with LVH strain pattern, 7 patients were male and 6 patients were female.

8. AGE WISE DISTRIBUTION OF LVH

LVH	NUMBER OF PATIENTS ≥ 50 yrs of Age	NUMBER OF PATIENTS< 50 yrs of age	TOTAL
WITHOUT STRAIN	18	13	31
WITH STRAIN	7	5	12
TOTAL	25	18	43

The incidence of LVH in patients greater than 50 years of age was 18.9% and it was 15.2% in patients lesser than 50 years of age.Out of the 31 patients with LVH without strain, 18 patients belonged to ≥ 50 years of age group and 13 patients were from < 50 years age group. Out of the 12 patients with LVH strain pattern, 7 patients were in ≥ 50 yrs of age group and 5 patients in < 50 years of age. 25 patients in ≥ 50 yrs age group had LVH and 18 patients in < 50 years age group had LVH.

9. **CORRELATION BETWEEN LVH AND DIASTOLIC BP**

LVH	**PATIENTS WITH DIASTOLIC BP**		**TOTAL**
	< 110 MM Hg	**≥110 mmHg**	
WITHOUT STRAIN	**11**	**20**	**31**
WITH STRAIN	**2**	**10**	**12**
TOTAL	**13**	**30**	**43**

Out of the 31 patients with LVH without strain, 20 patients had diastolic BP ≥ 110 mm Hg and 11 patients had diastolic BP < 110 mm Hg. Out of the 12 patients with LVH strain pattern 10 patients had diastolic BP ≥ 110 mm Hg and 2 patients had diastolic BP < 110 mm Hg .

30 patients with diastolic BP ≥ 110 mm Hg had LVH and 13 patients with diastolic BP < 110 mm Hg had LVH.

10. CORRELATION BETWEEN LVH AND IVCD

	RBBB	**LAFB**
NO. OF PATIENTS WITH LVH	1	7

In the group of 43 patients with LVH, 7 of them had associated LAFB and 1 patient had RBBB.

DISCUSSION

From the results it is evident that intraventricular conduction disturbances and left ventricular hypertrophy form one of the main ECG changes in hypertension. The occurrence of intraventricular conduction disturbances and left ventricular hypertrophy as a part of cardiovascular complications of hypertension is well known. The Framingham study has shown that the incidence of left ventricular hypertrophy and conduction disturbances is higher among hypertensives.

INTRAVENTRICULAR CONDUCTION DISTRUABNCES

A review of hypertension and left ventricular hypertrophy by Franz.H.Messerlt et al, states that the increased incidence of conduction disturbances in hypertensives is probably due to the increased fibrous tissue or altered collagen content [52]

Martin et al in their recent electrophysiological studies have demonstrated that conduction disturbances develop in hypertrophied ventricles in the presence of myocardial ischaemia .[53] many studies have proved the presence of coronary ischaemia in spite of normal coronary arteries in hypertension

While describing about left ventricular hypertrophy as one of the causes of left anterior fascicular block, Goldman suggests, “Abnormal degree of left axis

deviation is probably not due to the hypertrophied mass as such, but to the associated subendocardial fibrosis involving the anterior fascicle of the left bundle.[54]

Gopinath et al, in their three year follow up study of hypertension in Delhi recorded ECG's of 1417 patients, out of which 237 patients (16.7%) had LVH with or without strain and 110 patients (7.8%) had intraventricular conduction disturbances. [55]

In our study, the incidence of intraventricular conduction disturbances was slightly higher (10%) as opposed to 7.8% in the above study. The incidence of LVH with or without strain in our study was 17.2% and it was 16.7% in the above study. The results of our study correlates well with the above study.

While comparing the relative incidences of various conductions defects in our study population, LAFB (7.2%) was more frequently observed than RBBB (2.4%) and LBBB (0.4%). This could be explained by the fact that the right bundle and the anterior fascicle of the left bundle are long thread like structures. In addition, the anterior fascicle of left bundle passes below the aortic valve in the left ventricular outflow tract, which is an area of high flow velocity and receives its blood supply from only one vessel, the left anterior descending coronary artery. The main left bundle divides closer to its origin than the right

bundle, so that a relatively smaller area of the left bundle is vulnerable to inferference. Hence hypertensive heart disease is more likely to involve the left anterior fascicle and the right bundle, which was confirmed by our study.

LPFB was not observed in our study population. The compactness of the left posterior fascicle makes it the least vulnerable segment of the whole intraventricular conduction system.

SEX AND AGE DISTRIBUTION

While considering the relative incidences of various conduction defects in male and female hypertensives, it was 10.7% and 9.1% respectively. This indicates that conduction defects were observed almost equally in both sexes.

When patients were arbitrarily divided into two groups based on their age such as age ≥ 50 yrs and age < 50 years, the relative incidences of intraventricular conduction defects were 12.3% and 6.7% respectively. The increased incidence of conduction defects in older patients could be explained by the age related degeneration of the conduction system in addition to the subendocardial fibrosis induced by the hypertensive heart disease.

Irrespective of the type of conduction disturbance, most of these patients had increased diastolic BP (≥ 110 mmHg) It was found that out of the 18 patients with LAFB, 13 of them had high BP. This suggests that a high diastolic BP

leads to conduction disturbance as LVH and myocardial ischaemia secondary to hypertensive heart disease are common in patients with high blood pressure.

LEFT VENTRICULAR HYPERTROPHY

SEX AND AGE DISTRIBUTION

The incidence of LVH in male and female hypertensives were 15.3% and 19.1% respectively. This clearly demonstrates that female hypertensives were slightly more prone to develop LVH. Several epidemiological studies on hypertension has shown increased incidence of LVH in females.[49,50,56]

The incidence of LVH in patients greater than 50 yrs of age and lesser than 50 yrs of age were 18.9% and 15.25% respectively. This shows that LVH is more likely to be observed in hypertensives who were older. In this regard. Simone et al in his study of evaluation of concentric LVH in humans reports that the prevalence of LVH rises as age increases. [57]

Among the 43 patients with LVH, a majority of them (30 patients) had diastolic BP greater than ≥ 110 mmHg. This shows that the likelihood of developing LVH increases with high blood pressures. This has been demonstrated in various studies. [51]

Among the 43 patients with LVH, it was observed that 7 of them had associated LAFB and 1 patient had RBBB.

Though an objective study was made regarding the number of years of hypertension and the occurrence of conduction disturbances and LVH, no decisive interference could be taken because many patients were aware of having hypertension only from the day they visited a doctor for some other complaint. Hypertension was accidentally detected by them. While some patients had symptoms referable to hypertension, they were having indigenous treatment or postponing consultation with a doctor for an indefinite period. So the exact duration of hypertension in them could not be assessed.

CONCLUSION

This study entitled “The study of intraventricular conduction disturbances and left ventricular hypertrophy in systemic hypertension” included the study of 250 patients of systemic hypertension with the clinical presentation and ECG findings. The following conclusions were derived.

1. The incidence of intraventricular conduction disturbance in systemic hypertension was 10% .
2. LAFB was the most common conduction disturbance in systemic hypertension followed by RBBB and LBBB. LPFB was not observed
3. The distribution of conduction disturbances were almost equal in males and females.
4. Conduction disturbances were more common in the older age group.
5. The incidence of LVH in systemic hypertension was 17.2% .
6. The incidence of LVH without strain and with strain were 12.4% and 4.8% respectively.
7. Female hypertensives were more likely to have LVH .
8. LVH was more common in the older age group.
9. Patients with conduction disturbances and LVH were more likely to have high BP.
10. The presence of intraventricular conduction disturbances and LVH in hypertensives indicates the need for more vigilant antihypertensive therapy.

PROFORMA

NAME AGE SEX

OCCUPATION

HYPERTENSION CLINIC NUMBER

DATES OF EXAMINATION

DURATION OF DETECTION OF HYPERTENSION

GENERAL EXAMINATION

- BUILT, NOURISHMENT
- PALLOR
- ICTERUS
- CYANOSIS
- CLUBBING
- PEDAL EDEMA

VITALS

- PULSE- RATE, RHYTHM, CHARACTER
- BLOOD PRESSURE- RIGHT ARM, LEFT ARM
- RESPIRATORY RATE
- TEMPERATURE

EXAMINATION OF CARDIOVASCULAR SYSTEM

CHESTWALL SYMMETRY

APICAL IMPULSE , THRILL, PALBABLE SOUNDS

FIRST HEART SOUND - NORMAL / LOUD / SOFT

SECOND HEART SOUND - NORMAL / LOUD/ SOFT / SPLIT

MURMUR, RUB

RESPIRATORY SYSTEM

BREATH SOUNDS

ADDED SOUNDS

ABDOMEN

ORGANOMEGALY

FREE FLUID

NEUROLOGICAL EXAMINATION

INVESTIGATIONS

ELECTROCARDIOGRAM

- RATE, RHYTHM
- QRS – AXIS, DURATION, CONFIGURATION
- VENTRICULAR ACTIVATION TIME
- CHAMBER ENLARGEMENT
- ST-T CHANGES

BLOOD SUGAR

BLOOD UREA

SERUM CREATININE

SERUM CHOLESTEROL

URINE ROUTINE

CHEST X-RAY PA VIEW

TREATMENT

MASTER CHART

SNO	NAME	AGE	SEX	DURATION OF HYPERTENSION	BP mm Hg	RATE	RHYTHM	QRS DURATION (ms)	QRS AXIS	QRS CONFIGURATION	ST-T CHANGES	COMMENT
1	SHANTHI	40	F	2	170/100	70	NSR	0.06	+30°	N	-	N
2	RANI	45	F	2	140/90	80	NSR	0.08	+45°	N	-	N
3	MADANAGOPAL	65	M	4	190/120	98	NSR	0.06	+15°	RV_5 = 3.2 mv	-	LVH
4	LEELA DEVI	76	F	2	150/90	72	NSR	0.08	+45°	N	*	N
5	SAROJA	48	F	3	150/100	80	NSR	0.10	+60°	N	-	N
6	PACHIAMMAL	50	F	3	160/90	90	NSR	0.06	+30°	N	-	N
7	RAMACHANDRAN	51	M	6	150/100	76	NSR	0.10	-60°	qR in LI, aV_L rs in LII, III, a Vf	-	LAFB
8	RAJENDRAN	35	M	3	140/90	80	NSR	0.08	+15°	N	-	N
9	SANKAR	48	M	3	160/100	78	NSR	0.06	+30°	N	-	N
10	MARIAMMAL	65	F	3	160/100	94	NSR	0.08	-60°	qR in LI, aV_L rs in LII, III, aVf ; RV_6 = 3 mv	-	LVH; LAFB
11	PALANI	50	M	4	170/90	82	NSR	0.08	+45°	N	-	N
12	ABDUL	70	M	3	160/100	74	NSR	0.06	+60°	N	-	N
13	GOVINDARAJ	70	M	6	180/114	90	NSR	0.14	-45°	RR^1 IN V_6 ; Wide S IN V_1 V_2	TWAVE (v_5.v_6)	LBBB
14	JINNA	47	M	2	150/100	76	NSR	0.08	+15°	R V6 = 3 mv	-	LVH
15	CHINNAKUTTY	75	M	2	160/100	84	NSR	0.06	+60°	N	-	N
16	MUNIAMMAL	46	F	2	180/110	86	NSR	0.06	+30°	SV_1 + RV 5 = 4.2 mv	-	LVH
17	SHANMUGAM	50	M	4	160/100	94	NSR	0.08	+15°	N	-	N
18	BABU	53	M	2	150/100	76	NSR	0.08	+30°	N	-	N
19	SRINIVASAN	32	M	3	160/100	86	NSR	0.06	+60°	N	-	N
20	ARUMUGAN	55	M	4	160/90	68	NSR	0.08	+30°	N	-	N

21	ANNALAKSHMI	58	F	3	160/104	80	NSR	0.14	+20°	rSR ' in V_1 ; wide S in V_{4-6}	-	RBBB
22	CHAKUBAI	60	F	4	150/90	72	NSR	0.06	+60°	N	-	N
23	THERESA	40	F	3	140/100	80	NSR	0.08	+45°	N	-	N
24	RANIAMMAL	58	F	2	160/100	82	NSR	0.08	+45°	N	-	N
25	SHANTHA DEVI	70	F	3	190/110	76	NSR	0.14	+20°	rSR' in V_1 V_2 V_3	T WAVE V_1 V_2	RBBB
26	MALLIKABEE	52	F	2	160/90	90	NSR	0.10	+30°	N	-	N
27	ALADDIN	57	M	4	150/96	80	NSR	0.06	+15°	RV 5 = 3 mv	ST V_{4-6}	LVH WITH STRAIN
28	KHAJAMOHAMED	42	M	3	160/90	94	NSR	0.06	+15°	N	-	N
29	JANAKI	48	M	2	170/94	96	NSR	0.06	+30°	N	-	N
30	RAMESH	52	M	6	170/96	74	NSR	0.10	+45°	N	-	N
31	BASKAR	66	M	3	150/90	68	NSR	0.06	+30°	N	-	N
32	MARIADOSS	70	M	3	170/114	84	NSR	0.10	-45°	qR In LI, avl; rS in LII, III, a Vf	-	LAFB
33	KESAVAN	56	M	4	170/100	76	NSR	0.08	+45°	N	-	N
34	RAJAGOPAL	60	M	3	170/100	68	NSR	0.06	+30°	N	-	N
35	SHANMUGAM	50	M	4	150/0	78	NSR	0.08	+15°	N	-	N
36	KALIAMMAL	45	F	2	150/90	84	NSR	0.06	+30°	N	-	N
37	AHMED	52	M	6	180/120	70	NSR	0.08	-30°	qR in L I, $AV_{L;}$ rS in LII, III, Avf	-	LAFB
38	THARA	40	F	2	150/90	90	NSR	0.06	+45°	N	-	N
39	JAMALYA BEGUM	45	F	3	190/118	80	NSR	0.06	+30°	SV1 + RV6 = 4 mv	-	LVH
40	RAJA	50	M	2	160/100	92	NSR	0.08	+60°	N	-	N
41	VEMBULI	70	F	2	180/90	90	NSR	0.08	+30°	N	-	N
42	KANNIAPPAN	76	M	4	170/90	78	NSR	0.08	+45°	N	-	N
43	KANNAYIRAM	58	M	2	130/90	80	NSR	0.06	+30°	N	-	N
44	THAMSEEN	49	F	2	150/90	82	NSR	0.06	+45°	N	-	N
45	SAKUNTHALA	60	F	3	170/90	84	NSR	0.08	+30°	N	-	N

46	ANNAKILI	65	F	3	140/90	82	NSR	0.08	+45°	N	-	N
47	NARAYANAN	65	M	4	160/100	92	NSR	0.06	+30°	N	-	N
48	RADHA	73	F	4	174/116	80	NSR	0.06	-30°	qR in LI, aVL, rs in LII, III, a VF	-	LAFB
49	MURUGAN	42	M	3	180/100	70	NSR	0.08	+45°	N	-	N
50	PAPATHIAMMAL	56	F	3	150/100	78	NSR	0.10	+30°	N	-	N
51	NITHYANANDAM	48	M	3	160/100	76	NSR	0.06	+30°	R V6=3 mv	-	LVH
52	LINGAM	49	M	2	150/90	74	NSR	0.06	+45	N	-	N
53	PUNITHAM	60	F	2	180/114	90	NSR	0.06	-15°	SV1 +RV6 = 4.2 mv	-	LVH
54	JOHN	36	M	3	150/90	92	NSR	0.08	+15°	N	-	N
55	FATHIMA	45	F	4	160/90	96	NSR	0.06	+30°	N	-	N
56	RAJKUMAR	40	M	3	130/90	78	NSR	0.08	+45°	N	-	N
57	ABDUL JAFFER	60	M	5	170/112	92	NSR	0.06	-45°	qR IN LI, Avl; rs in LII, III, aVf	-	LAFB
58	SANKARAN	59	M	4	140/90	88	NSR	0.06	+30°	N	-	N
59	RATHNA BAI	60	F	2	180/90	82	NSR	0.08	+45°	N	-	N
60	SATHIK ALI	50	M	2	160/90	76	NSR	0.06	+15°	N	-	N
61	KABALI	55	M	3	200/120	80	NSR	0.06	+10°	R V5 = 3.4 mv	ST V_{4-6}	LVH WITH STRAIN
62	MAHMOOD	45	M	4	160/90	68	NSR	0.10	+30°	N	-	N
63	SARADHA	70	F	4	160/100	62	NSR	0.08	+45°	N	-	N
64	CHARLES	77	M	7	150/100	78	NSR	0.10	+15°	N	-	N
65	MANIKUMAR	40	M	3	150/90	86	NSR	0.06	+30°	N	-	N
66	ABDUL SALAAM	52	M	6	180/120	78	NSR	0.08	-45°	qR in LI, aVl; rs in LII, III avf ; RV6 = 3.2 mv	-	LVH ; LAFB
67	SOUNDARAM	45	F	3	170/90	68	NSR	0.06	+45°	N	-	N
68	SULAIMAN	32	M	2	150/90	74	NSR	0.06	+10°	N	-	N
69	JANAKI	45	F	3	170/90	72	NSR	0.06	+30°	N	-	N

70	SULOCHANA	48	F	4	190/120	66	NSR	0.08	+15°	SV1 + RV5 = 4 mv	-	LVH
71	MEENA	40	F	3	150/94	68	NSR	0.08	+45°	N	-	N
72	AMARNATH	44	M	2	200/110	80	NSR	0.14	+5°	rSR' in V1 V2	TWAVE V1 V2	RBBB
73	SUGUNESHWARI	58	F	4	150/90	84	NSR	0.10	+15°	N	-	N
74	RAJAJI	34	M	2	200/116	72	NSR	0.06	+15°	R V6 = 3 mv	ST V4-6	LVH with strain
75	YASODHA	60	F	4	150/100	76	NSR	0.06	+30°	N	-	N
76	JANIBEE	44	F	3	160/94	80	NSR	0.08	+45°	N	-	N
77	KUTTYRAJ	36	M	3	156/100	90	NSR	0.06	-15°	R V5 = 3.2 mv	-	LVH
78	MUMTAZ	46	F	4	160/94	98	NSR	0.08	+30°	N	-	N
79	ADHILAKHSMI	44	F	2	130/94	94	NSR	0.06	+45°	N	-	N
80	ALAMELU	60	F	2	150/90	78	NSR	0.08	+60°	N	-	N
81	RANI	50	F	5	146/92	90	NSR	0.08	-60°	qR in LI, avl; rS in LII, III avf	-	LAFB
82	BABU	44	M	3	150/90	84	NSR	0.10	+45°	N	-	N
83	SILAMBARASAN	30	M	2	140/90	82	NSR	0.08	+15°	N	-	N
84	RAJENDRAN	43	M	3	144/80	90	NSR	0.06	+60°	N	-	N
85	ANGAMMAL	52	F	3	210/120	82	NSR	0.06	-15°	R V5 = 3.2mv	ST V4-6	LVH with strain
86	SHEIK DAWOOD	42	M	2	140/90	74	NSR	0.08	+30°	N	-	N
87	RAJ	52	M	4	170/90	76	NSR	0.06	+15°	N	-	N
88	ADAIKALAMARY	44	F	2	136/90	78	NSR	0.08	+45°	N	-	N
89	SANKARAN	48	M	3	170/90	84	NSR	0.08	+45°	N	-	N
90	LAKSHMI	40	F	2	130/90	86	NSR	0.08	+60°	N	-	N
91	CHINNAIH	50	M	3	160/80	64	NSR	0.06	+15°	N	-	N
92	MANI	65	M	3	150/90	72	NSR	0.06	+30°	N	-	N
93	DHANAM	40	F	2	160/90	62	NSR	0.08	+45°	N	-	N
94	HAZEERA BEGUM	42	F	6	170/130	80	NSR	0.10	-45°	qR IN LI, aVL; rS in LI, III, aVF	-	LAFB

95	CHANDRAN	57	M	2	160/90	74	NSR	0.08	+60°	N	-	N
96	VALLI	45	F	4	160/96	76	NSR	0.06	+5°	RV5 = 3 mv	ST V4-6	LVH strain
97	ABDUL GANI	37	M	3	140/84	82	NSR	0.10	+45°	N	-	N
98	PADMAVATHY	41	F	3	150/96	90	NSR	0.08	+30°	N	-	N
99	ANBALAGAN	52	M	2	200/120	80	NSR	0.06	-15°	S V1 +RV6 = 4 mv	-	LVH
100	DEVENDRAN	70	M	2	150/90	92	NSR	0.08	+45°	N	-	N
101	NOORJAHAN	45	F	3	190/116	66	NSR	0.06	+45°	RV6 = 3 mv	-	LVH
102	RUKMANI	60	F	6	170/90	68	NSR	0.08	+30°	N	-	N
103	RAJENDRAN	50	M	2	160/84	80	NSR	0.06	+15°	N	-	N
104	MOWLANA	53	M	3	180/116	90	NSR	0.14	+20°	rSR' in V1 V2 V3	TWAVE V1V2	RBBB
105	DHANALAKSHMI	40	F	4	150/92	70	NSR	0.06	+30°	N	-	N
106	GURUNATHAN	36	M	3	150/90	74	NSR	0.08	+10°	N	-	N
107	BABU	37	M	2	190/120	76	NSR	0.06	+30°	RV5=3.2 mv	ST V4-6	LVH WITH STRAIN
108	VEERARAGAVAN	60	M	4	150/90	92	NSR	0.06	+15°	N	-	N
109	DEVA	40	M	3	140/80	80	NSR	0.10	+10°	N	-	N
110	SURYA	46	M	3	140/80	80	NSR	0.08	+60°	N	*	N
111	SUSAIAMMAL	60	F	3	190/90	64	NSR	0.06	+30°	N	-	N
112	ILIYAS	50	M	4	144/80	68	NSR	0.10	+45°	N	-	N
113	SUGUNA	47	F	4	140/80	82	NSR	0.08	+30°	N	-	N
114	SUBRAMANI	35	M	3	140/90	92	NSR	0.06	+60°	N	-	N
115	RAMAN	35	M	3	150/90	70	NSR	0.06	+30°	N	-	N
116	PICHAMMAL	55	F	2	180/114	68	NSR	0.06	+30°	R V5,6 = 3 mv	-	LVH
117	NOORUDIN	53	M	3	160/100	74	NSR	0.08	+60°	N	-	N
118	PARTHASARATHY	44	M	4	140/90	86	NSR	0.10	+45°	N	-	N
119	SAKKUBAI	62	F	6	190/114	74	NSR	0.12	+5°	rSR' in V1; wide S in V4-6	-	RBBB

120	ABARANAVALLI	66	F	7	150/90	90	NSR	0.08	+15°	N	-	N
121	POONGAVANAM	72	F	7	144/92	94	NSR	0.06	+10°	N	-	N
122	BALAKRISHNAN	45	M	4	140/90	68	NSR	0.08	+60°	N	-	N
123	SHANTHAMA	40	F	2	146/92	70	NSR	0.10	+30°	N	-	N
124	DASTAGIR	70	M	4	160/100	80	NSR	0.10	-45°	qR in LI,aVl; rS in LII, LIII , aVF	-	LAFB
125	RAJESHWARI	45	F	4	150/80	94	NSR	0.06	+10°	N	-	N
126	INDRANI	63	F	S	162/90	82	NSR	0.08	+30°	N	-	N
127	RAMAJANEYULU	38	M	2	180/92	72	NSR	0.06	+30°	SV1 +RV5 = 4 mv	-	LVH
128	VALLIAMMAL	40	F	4	170/90	86	NSR	0.08	+60°	N	-	N
129	VIJAYALAKSHMI	50	F	5	144/80	76	NSR	0.10	+45°	N	-	N
130	ELUMALAI	70	M	6	180/110	80	NSR	0.08	-60°	qR in LI, aVL; rS in LII, III aVf ; RV6 = 3 mv	-	LVH, LAFB
131	JANAKI	63	F	5	180/110	68	NSR	0.06	+30°	N	-	N
132	RAHIM	55	M	4	160/90	84	NSR	0.06	+45°	N	-	N
133	LAKSHMI	40	F	4	154/80	92	NSR	0.08	+30°	N	-	N
134	VISALAKSHMI	52	F	5	160/90	96	NSR	0.08	+15°	N	-	N
135	MANI	48	M	3	180/120	70	NSR	0.08	-60°	qR in LI, aVL,;rS in LII, III aVF	-	LAFB
136	GNANAM	44	F	5	150/84	74	NSR	0.10	+30°	N	-	N
137	RAMARAJ	40	M	4	160/80	80	NSR	0.06	+45°	N	-	N
138	GHOUSIA BEGUM	55	F	8	180/114	72	NSR	0.06	-15°	SV1 +RV5 = 4 mv	ST V4-6	LVH WITH STRAIN
139	SOUNDARI	75	F	3	140/90	80	NSR	0.06	+30°	N	-	N
140	ANNAM	44	F	4	136/90	74	NSR	0.08	+45°	N	-	N
141	FATHIMA	70	F	6	144/92	76	NSR	0.10	+15°	N	-	N
142	NAGARAJ	43	M	2	170/100	90	NSR	0.08	+15°	RV6 = 3 mv	-	LVH
143	ARUMUGARAJ	45	M	4	130/90	92	NSR	0.08	+30°	N	-	N
144	KABIRUNISA	56	F	2	146/90	84	NSR	0.10	+60°	N	-	N

145	SELVAM	57	M	5	150/90	82	NSR	0.08	+45°	N	-	N
146	POWNAMMAL	45	F	2	180/114	90	NSR	0.08	-45°	qR in LI, Avl;rS in LII, III Avf; Rv5=3	-	LVH; LAFB
147	LEONARD BABU	37	M	2	140/86	78	NSR	0.06	+60°	N	-	N
148	JAYALAKSHMI	40	F	4	136/84	64	NSR	0.08	+30°	N	-	N
149	ASLAM BASHA	60	M	3	180/120	80	NSR	0.06	-15°	SV1 +RV5 = 4 mv	-	LVH
150	RAHMATHUNISA	48	F	4	170/90	66	NSR	0.10	+30°	N	-	N
151	RATHNAM	60	M	6	180/96	70	NSR	0.06	+40°	N	-	N
152	SARASWATHY	45	F	2	164/92	74	NSR	0.08	+10°	N	-	N
153	ANNAMALAI	68	M	4	150/100	76	NSR	0.14	+5°	rSR^1 in V1; RV5= 3.2 mv	-	LVH,RBBB
154	KRISHNAN	40	M	5	190/96	80	NSR	0.06	+15°	N	-	N
155	JAYA	60	F	6	144/80	82	NSR	0.08	+30°	N	-	N
156	ARJUN	30	M	2	166/90	84	NSR	0.06	+40°	N	-	N
157	HAMEED	65	M	3	170/92	86	NSR	0.08	+10°	N	-	N
158	SUSEELA	38	F	2	190/120	82	NSR	0.08	+30°	RV6 = 3.2 mv	-	LVH
159	SIVA	45	M	2	144/92	90	NSR	0.10	+45°	N	-	N
160	LAKSHMI	50	F	5	140/90	92	NSR	0.06	+30°	N	-	N
161	SIVARAMAN	45	M	3	160/92	76	NSR	0.08	+30°	N	-	N
162	NATARAJAN	65	M	5	140/90	74	NSR	0.06	+45°	N	-	N
163	SELVARANGAN	45	M	4	180/120	72	NSR	0.06	-30°	qR IN LI, aVL;rS in LII, III aVf	-	LAFB
164	KRISHNAKUMAR	65	M	6	160/90	82	NSR	0.08	+60°	N	-	N
165	VENKAT	47	M	4	140/80	84	NSR	0.06	+15°	N	-	N
166	ANWAR BASHA	60	M	5	180/114	86	NSR	0.08	+15°	SV1 +RV5 = 4.5 mv	ST V4-6	LVH WITH STRAIN
167	PRIYARANI	45	F	4	130/90	92	NSR	0.10	+30°	N	-	N
168	APARNA	36	F	3	134/84	90	NSR	0.06	+45°	N	-	N
169	RADHAKUMARI	48	F	4	160/90	88	NSR	0.08	+30°	N	-	N

170	INDRANI	42	F	4	134/84	74	NSR	0.06	+15°	N	-	N
171	LEELA	44	F	5	170/110	90	NSR	0.06	-45°	qR IN LI, aVL; rS in LII, III aVF	-	LAFB
172	CHELLAMMAL	45	F	4	180/120	80	NSR	0.08	+45°	R V5 = 3.4 mv	-	LVH
173	KUMAR	44	M	3	130/90	64	NSR	0.06	+30°	N	-	N
174	SWARNA	52	F	5	146/90	76	NSR	0.10	+15°	N	-	N
175	KARTHIK	40	M	4	150/90	82	NSR	0.08	+60°	N	-	N
176	ZAHIDHA	42	F	3	180/120	64	NSR	0.06	+30°	R↓V6 = 3.2 mv	ST V4-6	LVH WITH STRAIN
177	GOVIDNAN	65	M	6	150/92	96	NSR	0.08	+15°	N	-	N
178	VEERAKUMAR	66	M	7	140/90	92	NSR	0.06	+45°	N	-	N
179	MAHESH	52	M	5	150/94	68	NSR	0.08	+60°	N	-	N
180	NAGAMMAL	70	F	7	140/80	84	NSR	0.06	+60°	N	-	N
181	GOVIDNAMMAL	65	F	4	200/120	78	NSR	0.06	+60°	S V1 +R V5 = 4 mv	-	LVH
182	KAMALAKANNAN	47	M	3	144/90	60	NSR	0.10	+30°	N	-	N
183	RAMESWARI	46	F	4	160/92	72	NSR	0.06	+45°	N	-	N
184	RATHNA	44	F	3	144/90	76	NSR	0.06	+15°	N	-	N
185	RAMANKUTTY	65	M	5	150/100	80	NSR	0.10	-60°	qR in LI, aVL ;rS in LII, III aVF	-	LAFB
186	MARAGATHAM	60	F	6	154/90	78	NSR	0.08	+30°	N	-	N
187	ANNAMMAL	64	F	4	140/90	84	NSR	0.10	+45°	N	-	N
188	PARVATHY	60	F	3	180/116	70	NSR	0.06	+15°	SV1↓+RV6 = 4 mv	ST V 4-6	LVH WITH STRAIN
189	MEENAKUMARI	40	F	4	140/92	64	NSR	0.06	+15°	N	-	N
190	JAYAPANDIAN	68	M	2	150/110	68	NSR	0.08	+30°	N	-	N
191	KRISHNAVENI	60	F	6	200/100	80	NSR	0.06	+15°	R V6 = 3.2 MV	-	LVH
192	KASINATHAN	46	M	4	136/92	90	NSR	0.06	+60°	N	-	N
193	RAMESH	54	M	3	140/90	92	NSR	0.08	+10°	N	-	N
194	RAMANATHAN	42	M	4	150/92	72	NSR	0.06	+15°	N	-	N

195	ELLAMMAL	57	F	3	172/114	76	NSR	0.08	-45°	qR in LI,Avl;rS in LII, III, aVF	-	LAFB
196	SUGAVANAM	46	F	4	130/84	74	NSR	0.08	+30°	N	-	N
197	KASIAMMAL	60	F	4	160/90	84	NSR	0.10	+45°	N	-	N
198	SRIRANGAN	50	M	6	210/120	90	NSR	0.06	+15°	R V6 = 3 mv	-	LVH
199	NARAYANAN	50	M	5	130/90	92	NSR	0.08	+15°	N	-	N
200	LALITHA	44	F	4	144/92	96	NSR	0.06	+30°	N	-	N
201	JAYAM	46	F	3	130/90	68	NSR	0.10	+45°	N	-	N
202	KUMARAN	50	M	4	160/92	88	NSR	0.06	+60°	N	-	N
203	BALAN	40	M	3	136/82	90	NSR	0.08	+30°	N	-	N
204	SENTHAMARAI	60	F	6	180/116	68	NSR	0.08	+45°	SV1 +RV6 = 4.2 mv	-	LVH
205	SHYAMALA	40	F	3	140/92	72	NSR	0.06	+45°	N	-	N
206	SAVITHRI	52	F	5	160/90	74	NSR	0.10	+15°	N	-	N
207	RAJAMBAL	60	F	5	169/90	70	NSR	0.06	+30°	N	-	N
208	YOGAM	50	F	6	146/92	92	NSR	0.08	+15°	N	-	N
209	RAJESWARI	70	F	3	170/100	90	NSR	0.08	-45°	qR in LI, aVL; rS in LII, III, aVF	-	LAFB
210	GANESH	39	M	2	140/84	82	NSR	0.10	+15°	N	-	N
211	YOGANANTHAN	68	M	3	200/110	80	NSR	0.06	+15°	SV1+RV6=4 mv	-	LVH
212	BALARAMAN	52	M	4	150/90	74	NSR	0.08	+45°	N	-	N
213	PANEER	54	M	3	134/90	84	NSR	0.08	+45°	N	-	N
214	RAJAM	65	F	3	150/100	76	NSR	0.08	+15°	SV1 +RV6=4.1mv	-	LVH
215	SELVAM	44	M	4	160/94	78	NSR	0.10	+45°	N	-	N
216	JEYAKANTHAN	46	M	3	152/92	86	NSR	0.06	+60°	N	-	N
217	PADMA	44	F	4	136/90	64	NSR	0.08	+30°	N	-	N
218	PUSHPAGANDHI	40	F	8	180/120	74	NSR	0.10	-30°	qR LI aVL ; rS LII, III aVF	-	LAFB
219	SHIVAM	60	M	7	154/90	62	NSR	0.06	+5°	N	-	N

220	SHIVAKUMAR	44	M	2	168/88	86	NSR	0.08	+15°	N	-	N
221	KOMALAM	52	F	5	154/86	76	NSR	0.06	+30°	N	-	N
222	ANJALAI	52	F	6	200/120	82	NSR	0.06	+30°	R V5=3 mv	ST V 4-6	LVH WITH STRAIN
223	MURUGAN	54	M	5	150/84	96	NSR	0.08	+45°	N	-	N
224	MALA	38	F	4	168/88	68	NSR	0.06	+60°	N	-	N
225	RAJU	60	M	7	170/90	86	NSR	0.08	+15°	N	-	N
226	VENKATESAN	60	M	6	170/90	68	NSR	0.10	+30°	N	-	N
227	BANUBEE	68	F	3	180/110	90	NSR	0.06	-15°	RV6 = 3.2 mv	-	LVH
228	ABDUL KARIM	50	M	3	136/90	84	NSR	0.08	+45°	N	-	N
229	SANGLIMURUGAN	46	M	4	150/94	90	NSR	0.06	+30°	N	-	N
230	RASAAITHI	44	F	3	136/92	80	NSR	0.06	+30°	N	-	N
231	SANKAR	71	M	4	180/114	78	NSR	0.08	-60°	qR LI, aVL ; rS LII III aVF	-	LAFB
232	MUNIYANDI	60	M	6	160/90	86	NSR	0.08	+15°	N	-	N
233	SUBRAMANI	67	M	6	180/100	80	NSR	0.06	-45°	rS LII, III aVF ; RV6 = 3 mv	-	LVH, LAFB
234	KOKILA	46	F	3	140/90	72	NSR	0.06	+30°	N	-	N
235	ZAHIRA BHEE	45	F	3	160/96	78	NSR	0.08	+30°	SV1 + RV6 = 4 mv	-	LVH
236	SIVAKAMI	47	F	4	144/90	64	NSR	0.10	+60°	N	-	N
237	KUMARESAN	38	M	3	150/92	68	NSR	0.08	+60°	N	-	N
238	JAYARAMAN	44	M	4	150/100	76	NSR	0.14	+20°	rS R' in V1,V2, V3	TWAVE IN V1 V2	RBBB
239	KILIAMMAL	54	F	5	140/90	74	NSR	0.06	+30°	N	-	N
240	RAMESHWARI	52	F	4	162/96	92	NSR	0.08	+45°	N		N
241	AROCIAMMAL	70	F	3	200/114	80	NSR	0.08	-45°	qR LI, aVL : rS LII III aVF ; RV5 = 3 mv	-	LVH
242	SIVANANDI	80	M	7	160/92	66	NSR	0.06	+15°	N	-	N
243	MUMTAJ	44	F	3	140/100	96	NSR	0.06	+60	N	-	N

244	KAMALAM	54	F	4	154/92	70	NSR	0.08	+30°	N	-	N
245	SUNDARAMA	60	F	3	170/120	64	NSR	0.10	-45°	qR LI, aVL ; rS LII III aVF	-	LAFB
246	SAGAYAMARI	44	F	4	136/94	72	NSR	0.06	+30°	N	-	N
247	MOHAMMED	60	M	5	180/120	82	NSR	0.06	-60°	qR LI, aVL ; rS LII III aVF	-	LAFB
248	SWAMINATHAN	62	M	7	130/90	76	NSR	0.08	+15°	N	-	N
249	NARASIMHAN	40	M	5	170/110	78	NSR	0.06	+30°	SV1 + RV5 = 4.2 mv	ST V4-6	LVH WITH STRAIN
250	SIVANESAN	64	M	6	160/92	80	NSR	0.10	+30°	N	-	N

KEY TO MASTER CHART

NSR	**-**	**NORMAL SINUS RHYTHM**
M	**-**	**MALE**
F	**-**	**FEMALE**
LVH	**-**	**LEFT VENTRICULAR HYPERTROPHY**
LAFB	**-**	**LEFT ANTERIOR FASCICULAR BLOCK**
RBBB	**-**	**RIGHT BUNDLE BRANCH BLOCK**
LBBB	**-**	**LEFT BUNDLE BRANCH BLOCK**
N	**-**	**NORMAL**
ms	**-**	**Milli Second**
mv	**-**	**Milli Volt**

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